

THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING

a report of the Surgeon General

1986

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
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parents smoke, a stronger relationship exists than if only one parent smokes.

What future respiratory burden these findings may represent for these children later in life is not known. As a former pediatric surgeon, I strongly urge parents to refrain from smoking in the presence of children as a means of protecting not only their children's current health status but also their own.

Diseases Other Than Lung Cancer

Several studies have provided data on the relationship between ETS and cancers other than lung cancer and on ETS exposure and cardiovascular disease. However, further research in these areas will be required to determine whether an association exists between ETS exposure and an increased risk of developing these diseases.

Policies Restricting Smoking in Public Places

The growth in our understanding of the disease risk associated with involuntary smoking has been accompanied by a change in the social acceptability of smoking and by a growing body of legislation, regulation, and voluntary action that addresses where smoking may occur in public. Forty States and the District of Columbia now have some form of legislation controlling or restricting smoking in various public settings. Some States limit smoking to only a few designated areas; however, States are increasingly developing and implementing comprehensive legislation that restricts smoking in many public settings, including the workplace. Nine States have restrictions that cover smoking not only by public employees but also by employees in the private sector.

No systematic evaluation of the effects these measures may have on smoking behavior has been conducted, but there is little doubt that strong public sentiment exists for implementing such restrictions. A number of national surveys conducted by voluntary health organizations, government agencies, and even the tobacco industry have documented that an overwhelming majority of both smokers and nonsmokers support restricting smoking in public.

Public Health Policy and Involuntary Smoking

The 1986 Surgeon General's Report on the Health Consequences of Involuntary Smoking clearly documents that nonsmokers are placed at increased risk for developing disease as the result of exposure to environmental tobacco smoke.

Critics often express that more research is required, that certain studies are flawed, or that we should delay action until more conclusive proof is produced. As both a physician and a public health

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Cigarette smoke is well established as a human carcinogen. The chemical composition of ETS is qualitatively similar to mainstream smoke and sidestream smoke and also acts as a carcinogen in bioassay systems. For many nonsmokers, the quantitative exposure to ETS is large enough to expect an increased risk of lung cancer to occur, and epidemiologic studies have demonstrated an increased lung cancer risk with involuntary smoking. In examining a low-dose exposure to a known carcinogen, it is rare to have such an abundance of evidence on which to make a judgment, and given this abundance of evidence, a clear judgment can now be made: exposure to ETS is a cause of lung cancer.

The data presented in this Report establish that a substantial number of the lung cancer deaths that occur among nonsmokers can be attributed to involuntary smoking. However, better data on the extent and variability of ETS exposure are needed to estimate the number of deaths with confidence.

Respiratory Disease

Acute and chronic respiratory diseases have also been linked to involuntary exposure to tobacco smoke; the evidence is strongest in infants. During the first 2 years of life, infants of parents who smoke are more likely than infants of nonsmoking parents to be hospitalized for bronchitis and pneumonia. Children whose parents smoke also develop respiratory symptoms more frequently, and they show small, but measurable, differences on tests of lung function when compared with children of nonsmoking parents.

Respiratory infections in young children represent a direct health burden for the children and their parents; moreover, these infections, and the reductions in pulmonary function found in the school-age children of smokers, may increase susceptibility to develop lung disease as an adult.

Several studies have reported small decrements in the average level of lung function in nonsmoking adults exposed to ETS. These differences may represent a response of the lung to chronic exposure to the irritants in ETS, but it seems unlikely that ETS exposure, by itself, is responsible for a substantial number of cases of clinically significant chronic obstructive lung disease. The small magnitude of the changes associated with ETS exposure suggests that only individuals with unusual susceptibility would be at risk of developing clinically evident disease from ETS exposure alone. However, ETS exposure may be a factor that contributes to the development of clinical disease in individuals with other causes of lung injury.

Cardiovascular Disease

A few studies have examined the relationship between involuntary smoking and cardiovascular disease, but no firm conclusion on

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the relationship can be made owing to the limited number of deaths in the studies.

Irritation

Perhaps the most common effect of tobacco smoke exposure is tissue irritation. The eyes appear to be especially sensitive to irritation by ETS, but the nose, throat, and airway may also be affected by smoke exposure. Irritation has been demonstrated to occur at levels that are similar to those found in real-life situations. The level of irritation increases with an increasing concentration of smoke and duration of exposure. In addition, participants in surveys report irritation and annoyance due to smoke in the environment under real-life conditions.

Determinants of Exposure

Exposure to ETS has been documented to be common in the United States, but additional data on the extent and determinants of exposure are needed to identify individuals within the population who have the highest exposure and are at greatest risk. Studies with biological markers and measurements of ETS components in indoor air confirm that measurable exposure to ETS is widespread. However, within exposed populations, levels of cotinine excretion and presumably ETS exposure vary greatly.

In a room or other indoor area, the size of the space, the number of smokers, the amount of ventilation, and other factors determine the concentration of tobacco smoke in the air. The technology for the cost-effective filtration of tobacco smoke from the air is not currently available, and because of their small size, the smoke particles remain suspended in the air for long periods of time; thus, the only way to remove smoke from indoor air is to increase the exchange of indoor air with clean outdoor air. The number of air changes per hour required to maintain acceptable indoor air quality is much higher when smoking is allowed than when smoking is prohibited.

Environmental tobacco smoke originates at the lighted tip of the cigarette, and exposure to ETS is greatest in proximity to the smoker. However, the smoke rapidly disseminates throughout any airspace contiguous with the space in which the smoking is taking place. Dissemination of smoke is not uniform, and substantial gradients in ETS levels have been demonstrated in different parts of the same airspace. The time course of tobacco smoke dissemination is rapid enough to ensure the spread of smoke throughout an airspace within an 8-hour workday. In the home, the presence of even one smoker can significantly increase levels of respirable suspended particulates.

These data lead to the conclusion that the simple separation of smokers and nonsmokers within the same airspace will reduce, but

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for parental smoking and childhood cancer is also not clear, and evaluation of this association is made difficult by the various definitions of exposure that have been used, including maternal and paternal smoking before, during, and after the pregnancy. Mothers and fathers who smoke during a pregnancy generally smoked before the conception and continue to smoke after the pregnancy. Thus, an effect of involuntary smoking after birth cannot readily be distinguished from genetic or transplacentally mediated effects.

Cardiovascular Diseases

A causal association between active cigarette smoking and cardiovascular disease is well established (US DHHS 1983). The relationship between cardiovascular disease and involuntary smoking has been examined in one case-control study and three prospective studies. In the case-control study by Lee and colleagues (1986), described previously, ischemic heart disease cases and controls did not show a statistically significant difference in their exposure to involuntary smoking, based on the smoking habits of spouses or on an index accounting for exposure at home, at work, and during travel and leisure. In the Japanese cohort study, Hirayama (1984b, 1985) reported an elevated risk for ischemic heart disease ($N=494$) in nonsmoking women married to smokers. The standardized mortality ratios when the husbands were nonsmokers, ex-smokers or smokers of 19 or more cigarettes per day, and smokers of 20 or more cigarettes per day were 1.0, 1.10, and 1.31, respectively (one-sided p for trend, 0.019).

In the Scottish followup study (Gillis et al. 1984), nonsmokers not exposed to tobacco smoke were compared with nonsmokers exposed to tobacco smoke with respect to the prevalence of cardiovascular symptoms at entry and mortality due to coronary heart disease. There was no consistent pattern of differences in coronary heart disease or symptoms between nonsmoking men exposed to tobacco smoke and their nonexposed counterparts. Nonsmoking women exposed to tobacco smoke exhibited a higher prevalence of angina and major ECG abnormality at entry, and also a higher mortality rate for all coronary diseases. However, rates of myocardial infarction mortality were higher for exposed nonsmoking men and women compared with the nonexposed nonsmokers. The rates were 31 and 4 per 10,000, respectively, for the nonexposed nonsmoking men and women, and 45 and 12 per 10,000, respectively, for the exposed nonsmoking men and women. None of the differences were tested for statistical significance.

In the Japanese and the Scottish studies, other known risk factors for cardiovascular diseases, i.e., systolic blood pressure, plasma cholesterol, were not accounted for in the analysis.

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In a study of heart disease, Garland and coworkers (1985) enrolled 82 percent of adults aged 50 to 79 between 1972 and 1974 in a predominantly white, upper-middle-class community in San Diego, California. Blood pressure and plasma cholesterol were measured at entry, and all participants responded to a standard interview that asked about smoking habits, history of heart disease, and other health-related variables. Excluding women who had a previous history of heart disease or stroke or who had ever smoked, 695 currently married nonsmoking women were classified by their husbands' self-reported smoking status at enrollment. After 10 years of followup, there were 19 deaths due to ischemic heart disease; the age-standardized mortality rates for nonsmoking wives whose husbands were nonsmokers, ex-smokers, and current smokers were 1.2, 3.6, and 2.7, respectively (one-sided p for trend, ≤ 0.10). After adjustment for age, systolic blood pressure, total plasma cholesterol, obesity index, and years of marriage, the relative risk for death due to ischemic heart disease for women married to current or former smokers at entry compared with women married to never smokers was 2.7 (one-sided $p \leq 0.10$).

The study's findings are not convincing from the point of view of sample stability. The total number of deaths due to ischemic heart disease was small, and the denominator in the relative risk calculation is unstable, based on the deaths of two women whose husbands had never smoked. Moreover, it is well established that the risk of coronary heart disease is substantially lower among those who have stopped smoking (US DHHS 1983), although the amount of time required for this change after cessation of smoking is not clear (Kannel 1981). In this study, 15 of 19 deaths occurred in nonsmoking women married to husbands who had stopped smoking at entry, and the age-standardized rate for ischemic heart disease was highest in this group. The high proportion of deaths in nonsmoking women married to men who became ex-smokers implies that the excess resulted from a sustained effect of involuntary smoking. More detailed characterizations of exposure to ETS and specific types of cardiovascular disease associated with this exposure are needed before an effect of involuntary smoking on the etiology of cardiovascular disease can be established.

One study (Aronow 1978a,b) suggested that involuntary smoking aggravates angina pectoris. This study was criticized because the end point, angina, was based on subjective evaluation, and because other factors such as stress were not controlled for (Coodley 1978; Robinson 1978; Waite 1978; Wakeham 1978). More important, the validity of Aronow's work has been questioned (Budiansky 1983).

Conclusions

1. Involuntary smoking can cause lung cancer in nonsmokers.
2. Although a substantial number of the lung cancers that occur in nonsmokers can be attributed to involuntary smoking, more data on the dose and distribution of ETS exposure in the population are needed in order to accurately estimate the magnitude of risk in the U.S. population.
3. The children of parents who smoke have an increased frequency of hospitalization for bronchitis and pneumonia during the first year of life when compared with the children of nonsmokers.
4. The children of parents who smoke have an increased frequency of a variety of acute respiratory illnesses and infections, including chest illnesses before 2 years of age and physician-diagnosed bronchitis, tracheitis, and laryngitis, when compared with the children of nonsmokers.
5. Chronic cough and phlegm are more frequent in children whose parents smoke compared with children of nonsmokers. The implications of chronic respiratory symptoms for respiratory health as an adult are unknown and deserve further study.
6. The children of parents who smoke have small differences in tests of pulmonary function when compared with the children of nonsmokers. Although this decrement is insufficient to cause symptoms, the possibility that it may increase susceptibility to chronic obstructive pulmonary disease with exposure to other agents in adult life, e.g., active smoking or occupational exposures, needs investigation.
7. Healthy adults exposed to environmental tobacco smoke may have small changes on pulmonary function testing, but are unlikely to experience clinically significant deficits in pulmonary function as a result of exposure to environmental tobacco smoke alone.
8. A number of studies report that chronic middle ear effusions are more common in young children whose parents smoke than in children of nonsmoking parents.
9. Validated questionnaires are needed for the assessment of recent and remote exposure to environmental tobacco smoke in the home, workplace, and other environments.
10. The associations between cancers, other than cancer of the lung, and involuntary smoking require further investigation before a determination can be made about the relationship of involuntary smoking to these cancers.
11. Further studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to

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determine whether involuntary smoking increases the risk of cardiovascular disease.

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